Ventricular septal defect caused by impact from a horseshoe

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Clinical Summary

A 16-year-old woman came to the emergency department 36 hours after a closed thoracic trauma that had occurred when she was dropped from her horse and then was trodden on by her horse. Acute dyspnea and concomitant hemoptysis were her reasons for seeking treatment. Physical examination showed a pulse of 130 beats/min and blood pressure of 95/45 mm Hg, paleness, and erosions over the skin in the image of a horseshoe. The heart had a normal S_1 and S_2 sounds and grade 6/6 holosystolic murmur at the left bottom sternal border irradiating to the right bottom sternal border. There was bibasal hypoventilation with a tubaric murmur on the right thorax. Electrocardiography demonstrated elevation of the ST segment in the right leads. Biochemical testing showed a hemoglobin level of 10.3 g/dL, total creatine kinase of 222 IU/L, MB isoenzyme of creatine kinase of 7.8 (index 3.5%), and cardiac troponin I level of 13.10 ng/mL. Chest radiography (Figure 1) showed a normal cardiac size, Kerley B lines, pulmonary congestion, and bilateral pleural effusion. Computed tomographic scan showed multiple rib fractures, pulmonary contusion, and pericardial effusion. These data were compatible with a ventricular septal defect. An echocardiogram was therefore performed (Figure 2). On transthoracic apical 4-chamber view, the ventricles appeared dilated and dysfunctional and demonstrated a ventricular septal

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Figure 1. Chest radiograph showing normal cardiac size, Kerley B lines up to middle lungs, bilateral pleural effusion with presence of venocapillary congestion, and prominent main pulmonary artery.

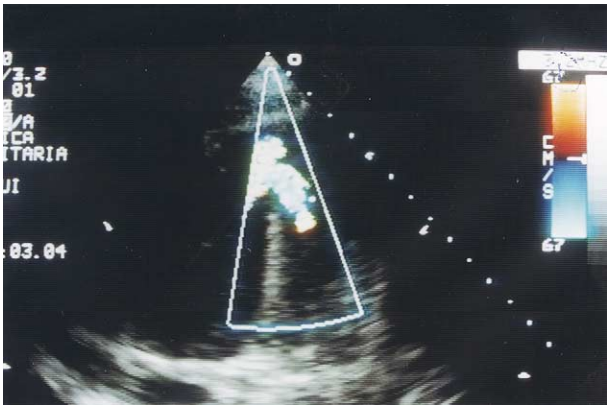

Figure 2. Transthoracic echocardiogram on apical 4-chamber view demonstrated ventricular septal defect 1.5 cm in length with significant left-to-right shunt.

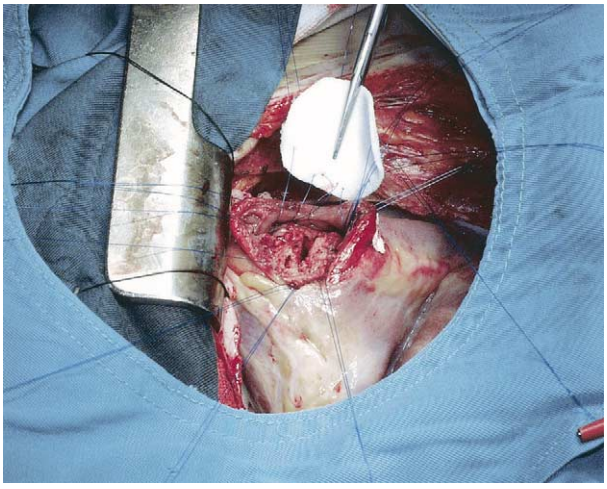

Figure 3. During surgical repair, ventricular septal defect was closed with 2-cm polytetrafluoroethylene patch.

defect 1.5 cm in length with a severe left-to-right shunt and wall motion abnormalities.

Inotropic support was started during the wait for surgical correction. Through a right ventricular approach, the ventricular septal defect was seen, with flattening of the flaps. In addition, there was severe dilatation of the right ventricle, with an hypokinetic area and a 3-cm contused region. The ventricular septal defect was closed with a 2-cm polytetrafluoroethylene patch (Figure 3). On echocardiography at discharge, there was no evidence of right or left ventricular dysfunction, and there were no signs of shunt. One year after the operation, the patient is free of symptoms and doing well.

Selective carotid cannulation at the neck: A satisfactory option for reoperation for aneurysms of the ascending aorta and arch

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We have evaluated the results of a modified Bachet technique in patients undergoing reoperation for aortic aneurysms. This technique was intended to reduce the risk of a cerebral dysfunction caused by prolonged deep hypothermic circulatory arrest[1] and bleeding caused by resternotomy.

Methods

Patients. From June 1999 to December 2001, a total of 4 patients underwent reoperation for ascending aortic or aortic arch aneurysm with selective cold cerebroplegia by bilateral cannulation of the common carotid arteries at the neck, as partially described by Bachet and colleagues.[2] A previous history of surgery on the ascending or aortic arch was present in all cases. A perianastomotic pseudoaneurysm was present at the proximal site in 1 patient and at the distal site in 3. One patient had a contained rupture of a proximal perianastomotic aortic aneurysm, with sternal erosion and expansion into the subcutaneous tissue (Figure 1, *A*).

Surgical technique. Common carotid arteries were exposed at the neck and cannulated with a 12F high-flow cannula without flange (Sarns-3M Health-Care, Borken, Germany; Figure 2, *A*). Cardiopulmonary bypass (CPB) was started by femoral cannulation, executing a Y junction along the arterial line to establish an antegrade flow, after replacement of the aortic arch (Figure 2, *B*). On reaching the desired temperature of 26°C, femoral perfusion was interrupted, common carotid arteries were proximally clamped, and the antegrade cerebral perfusion was accomplished by a centrifugal pump with independent line and heat-exchanger. A circulatory arrest of less than 3 minutes was induced at resternotomy, maintaining brain perfusion at a flow of 10 mL/(kg · min) and temperature at approximately 10°C.

Pressure into the perfusion line was established at 200 to 250 mm Hg, corresponding to 60 to 70 mm Hg in the carotid arteries, as described by Bachet and colleagues.[2] The ascending aorta and the arch were opened. The descending aorta was internally occluded with a Foley catheter, the subclavian artery was clamped to avoid steal phenomenon, and the CPB was restored to perfuse the lower half of the body at a flow greater than 500 mL/min. The left ventricle was vented through the right superior pulmonary vein. Antegrade intermittent cold blood cardioplegia was induced directly through the coronary ostia. When the distal aortic anastomosis was accomplished, we removed the Foley catheter, placed a proximal clamp on the vascular graft, vented the air, and reestablished the systemic antegrade blood flow through a side branch of a T-arm collagen-coated graft (Intergard; Hemabridge-Intervascular, La Ciotat, France). The extent of aortic replacement included ascending aorta and hemiarch replacement in 2 of the patients and complete ascending aorta and total arch replacement in the remaining 2, with 1 requiring a cuff of the epiaortic vessels and the other separate brachiocephalic and left carotid artery reimplantation (Figure 1, *B*).

The left subclavian artery was not involved in the aneurysm. The cerebral perfusion was interrupted, the carotid arteries were unclamped, and the carotid cannulas were removed. During the rewarming period we performed the proximal anastomosis and removed the graft clamp; at that time the air was completely vented and the CPB was progressively discontinued.

Results

The CPB time ranged between 89 and 125 minutes, whereas the antegrade cerebral perfusion time ranged between 37 and 76 minutes. One patient (25%) died of low cardiac output syndrome in the postoperative period. None of the patients had major neurologic dysfunction with evidence of focal or general deficit, and only 1 patient had a temporary neurologic dysfunction. A patient with excessive bleeding (1750 mL/36 hours) and requiring pro-

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